involved patients with complex disease and other mitigating factors. We therefore continue not only to use the connector but to promote its use to our colleagues.

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doi:10.1067/mtc.2003.282

Reply to the Editor:

Since submission of our report, we too have begun to administer clopidogrel routinely, 75 mg by mouth every morning starting on the first postoperative morning and continuing through the second postoperative month. We have detected no new problems.

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Symmetry aortic connector system

To the Editor:

The article by Donsky and colleagues[1] regarding thrombotic occlusion of vein grafts after use of the Symmetry aortic connector system (St Jude Medical, Inc, St Paul, Minn) aroused my interest because of my experience with this device. Similar to the authors' experience, I have had occlusion of the aortic orifice at the connector site within a few months of surgery in 3 patients. All patients were obese, diabetic, and hypertensive, as in the patients referenced in the article. It has been my experience, however, that these occlusions occur when a small (gray) connector is used, but not when a large (blue or purple) connector can be used. The authors do not mention the size of the connectors used in their case report. I would like to ask their opinion regarding my observation.

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doi:10.1067/mtc.2003.284

Reply to the Editor:

Size of the connector was not recorded in the permanent record.

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Does normothermic cardiopulmonary bypass influence clinical outcomes, cytokine production, and in vitro platelet function?

To the Editor:

We read with great interest the article by Gaudino and associates[1] in the June 2002 issue of this *Journal*. They evaluated prospectively the clinical outcomes of patients and some inflammatory and fibrinolytic markers such as C-reactive protein, interleukin (IL) 6, prothrombin time, and platelets. They concluded that normothermic systemic perfusion did not influence the clinical course or the extent of inflammatory and hemostatic activation in patients undergoing primary isolated coronary artery bypass. The markers were determined before surgical inetervention; 21, 48, and 72 hours thereafter; and at hospital discharge.

We[2] previously investigated platelet activation and aggregation up to 24 hours after operation by examining the serial changes of platelet count and small particle formation in patients undergoing coronary artery bypass with normothermic cardiopulmonary bypass (CPB). Platelet counts decreased during CPB, and 24 hours after CPB they had increased to approximately half the pre-CPB levels. Small particle formation was the main type of platelet aggregation observed before surgery. Medium particle formation was also recognized, but no patient had large particle formation. After systemic heparinization, small and medium particle formation occurred. One hour after the initiation of CPB, only small particles were seen; 2 hours after the end of CPB, no small particles were observed. Small particle formation was the main platelet aggregation type observed 24 hours after CPB.

We[3] also evaluated cytokine production and levels of thrombomodulin and soluble endothelium-derived adhesion molecules in patients undergoing coronary artery bypass under normothermic CPB. The study was scheduled also up to 24 hours after the operation. IL-6 values were elevated minimally after 30 minutes of CPB, and they showed a surge at the end of CPB or 2 hours after CPB in some patients. Other patients showed stable levels. The IL-6 values were reduced after 2 hours, but 24 hours after CPB they were still higher than the initial levels. There was a huge difference in IL-6 changes among patients. A surge of IL-8 occurred 2 hours after CPB, and the values returned to the initial levels 24 hours after CPB. Thrombomodulin levels were reduced 30 minutes after the initiation of CPB; however, they began to recover during CPB. The levels returned to the initial levels 2 hours after CPB and exceeded them 24 hours after CPB. Levels of soluble endothelium-derived adhesion molecules were reduced after 30 minutes of CPB; they returned to the initial levels 2 hours after CPB and exceeded them 24 hours after CPB.

Our studies may endorse the conclusions of Gaudino and associates. CPB for less than 3 hours does not influence clinical outcomes, and in vitro studies, such as platelet function and cytokine production, did not differ under normothermic and moderate hypothermic conditions.

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